Cardiac Cycle

Introduction

This lesson is about the flow of blood through the heart chambers and valves and the physiology of the heart chambers. The focus is on depicting the cardiac cycle from different perspectives.

You are going to see the cardiac cycle as heard by your ears, as felt by the left ventricle (pressure-volume loops), as experienced by the right atrium (by way of venous pulsations in the neck), and as seen from a normal ECG. Each perspective highlights very specific things. What is experienced in one can be approximated, inferred from the others. No one perspective provides a perfect mapping. Some depict time, and others don't. Some depict contraction or relaxation, and others don't. Some depict depolarization and repolarization, and others don't. Inference, approximation, and comparison will be made only to make sense of what's going on. This lesson is about mastering the cardiac cycle from multiple perspectives, not about tethering all those perspectives together. We do include the obligatory Wiggers diagrams (in Plumbing #3: *Valves*), which attempt to tether all those perspectives together—they are lies.

Systole can be defined as atrial or ventricular. It can be defined as chamber depolarization to repolarization (electrical systole). It can be defined as chamber contraction to relaxation (force systole). It can be defined as the period from the closure of the AV valves (S_1) to the closure of the semilunar valves (S_2; auscultated systole). In general, systole is when the chamber is active, acting, and doing things to other structures.

Diastole is all the time not spent in systole. Diastole is when the chamber is inactive, passive, and having things done to it.

We seek to define it in a number of ways.

Cardiac Cycle When You Listen

Heart sounds are made by the **closing of valves**. There are four valves for four chambers. They come in two sets. There are the **semilunar valves** (the valves that block artery-to-ventricle backflow) and the **atrioventricular valves** (the valves that block ventricle-to-atrium flow). The first heart sound (S_1) is made by the closing of the atrioventricular valves, and the second heart sound (S_2) is made by the closing of the semilunar valves. Each set of valves is grouped together because of the timing of their closure, the way we hear them with a stethoscope, and not because they represent the right or left side. Technically, there are four heart sounds, each sound made by the closing of one valve. In a normal patient without pathology, the atrioventricular valves close simultaneously, so their sounds are indistinguishable and collectively called S_1, whereas the semilunar valves close simultaneously, so their sounds are indistinguishable and collectively called S_2.

The cardiac cycle is divided into systole and diastole. The **duration of systole is fixed** and cannot be altered. The heart rate can vary greatly. Therefore, the **duration of diastole determines the heart rate** (this will be immensely important in the Electricity island). That means that S_1 and S_2 are separated by a fixed duration. The duration between S_2 and the next S_1 is determined by the heart rate, by the duration of diastole.

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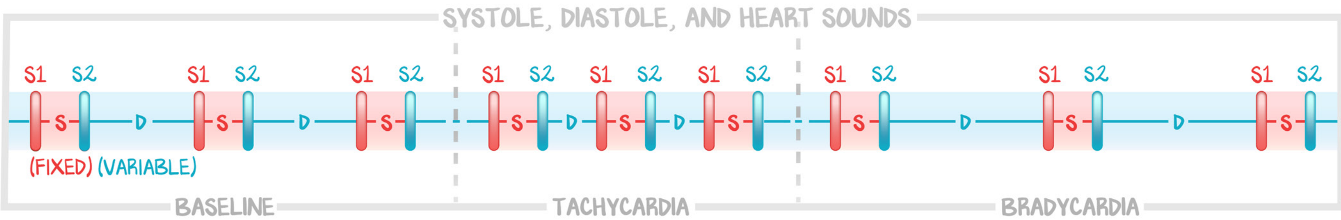

Figure 2.1: Systole, Diastole, and Heart Sounds
Systole is a fixed duration. The next systole (S_1–S_2) is determined by the heart rate. The heart rate is determined by the time spent in diastole. You cannot see the onset of depolarization, repolarization, contraction, filling, or ejection of blood. You can only hear the closure of the atrioventricular valves and then the closure of the semilunar valves and measure the time spent between them. When heart rates are fast and the time in diastole is short, it may be difficult to decide which is S_1 and S_2 by sound alone.

Cardiac Cycle as Seen From the Ventricle: Pressure-Volume Loops

The unidirectional flow of blood through the heart circuit is maintained by heart valves. The heart valves act as one-way doors, either opening forward into the next compartment or snapping shut to prevent the backflow of blood. The door opens when the pressure in the compartment behind the valve rises above the pressure in front of it. A valve closes when the pressure in the compartment behind it drops below the pressure of the compartment in front of it. When the valve opens, the pressures in the two chambers **equalize**. The **tricuspid** valve separates the right atrium from the right ventricle and opens into the right ventricle. The **pulmonic** valve separates the right ventricle from the pulmonary artery and opens into the pulmonary artery. The **mitral** valve separates the left atrium from the left ventricle and opens into the left ventricle. The **aortic** valve separates the left ventricle from the aorta and opens into the aorta.

We're going to tell the rest of this story using a two-chambered heart, using only the left atrium, left ventricle, and aorta. And we're going to do that with pressure-volume loops. There are four phases in the pressure-volume loop diagram, which we are going to list out of the order in which they occur—isovolumetric contraction, isovolumetric relaxation, ventricular filling, and ventricular ejection. There are isovolumetric [contraction/relaxation] and ventricular [filling/ejection]. When the ventricle begins to contract, enough pressure builds within the ventricle to close the mitral valve but not enough pressure to overcome the recoil force of the aorta. With both valves—aortic and mitral—closed, blood cannot go anywhere. If the blood (which is volume) doesn't move, the ventricle builds in pressure but doesn't change in volume—**isovolumetric contraction**. When the pressure in the ventricle exceeds the pressure in the aorta (systemic vascular resistance), the aortic valve opens, and blood is ejected from the ventricle. The pressure continues to build in the ventricle, but with a now open aortic valve, the blood leaves the ventricle into the aorta and arterial tree. Blood leaves the ventricle during **ventricular ejection**. At some point (that cannot be discerned on a pressure-volume loop), the ventricle repolarizes and relaxes. Blood continues to leave the ventricle until the relaxing heart, now with lowering pressure, reaches the point at which the aorta's recoil has a higher pressure than in the ventricle. When that happens, the aortic valve closes. With both the aortic and mitral valves closed, blood cannot move—the ventricle cannot fill with or empty of blood—which means that there is no change in volume—**isovolumetric relaxation**. When the pressure in the ventricle falls below the pressure in the atrium, the mitral valve opens, and blood begins to fill the ventricle—**ventricular filling**.

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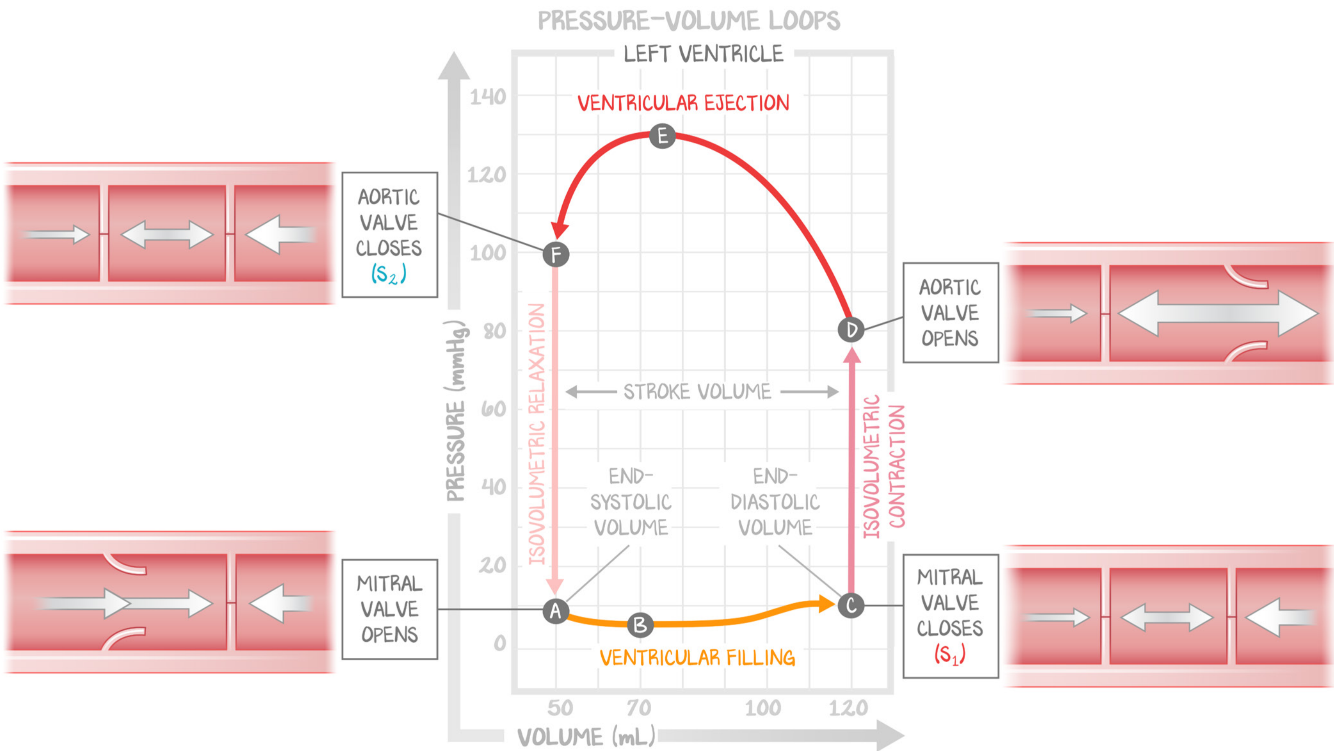

Figure 2.2: Pressure-volume Loops
Use this image to follow along with the text, phase 1–4, using the points and segments to track what's happening. Pressure is on the y-axis, and volume is on the x-axis.

We're going to break that last paragraph apart, adding what you've already learned about volumes (EDV, ESV, SV) and heart sounds. Whenever a **valve closes, it makes a sound**. Whenever a valve opens, it does not make a sound. The two heart sounds are S_1 (closure of the mitral valve) and S_2 (closure of the aortic valve). One can begin anywhere on a pressure-volume loop because it is a cycle. We chose to start at A in Figure 2.2, the bottom left of the loop. This is not about work, area under the curve, or changing parameters as we did in Structure and Function #5: *Myocardial Work*. It will be familiar, and it may even be redundant, engage it anyway as it is too crucial moving forward to miss.

Phase 1: Ventricular filling (points A, B, and C; segment AC). At position A, the aortic valve is already closed, and isovolumetric relaxation just occurred. As soon as the pressure in the atrium exceeds the pressure in the ventricle, the mitral valve opens (no sound), and filling begins. Initially (segment AB), the ventricular pressure decreases slightly because the ventricle has some give—it stretches in response to increased load. As volume increases, titin resists further distention of the ventricle, and passive tension develops, slightly increasing the pressure (segment BC). **Volume increases greatly, pressure increases slightly.**

The electrical impulse for a contraction happens. Diastole has ended, and systole has begun. The atrium depolarizes, then contracts, pushing the last bit of blood into the ventricle—the atrial kick. There is a pause at the AV node. The ventricle depolarizes. Then the ventricle contracts. You cannot see the electrical discharges on a pressure-volume loop, nor can you see the contraction and relaxation of the ventricle. You can only see the changes in pressure and volume and auscultate the closure of the valves.

Phase 2: Isovolumetric contraction (points C and D; segment CD). As the ventricle starts to contract, it generates pressure, active tension. That immediately exceeds the pressure in the left atrium, **closing the mitral valve** and generating the S_1 heart sound. The quintessential blood pressure is 120/80, a maximum of 120 at peak systole and 80 and minimum diastole. That is the same as saying that the ventricle can generate 120 mmHg and the aorta's recoil is as low as 80 mmHg. That also means that until the ventricle generates more than 80 mmHg, the aortic valve will remain closed, the backpressure keeping the aortic valve shut. So, during isovolumetric contraction, **both the mitral and aortic valves are closed**. Blood cannot move. If blood cannot move, the **volume stays the same**. This volume is the **end-diastolic volume** (EDV). As the ventricle generates active tension, as actin and myosin slide over one another, the **pressure increases**.

Think about blowing up a balloon. When you take the balloon out of the package, the balloon is open, but no air rushes out. Mentally inflate the balloon and hold it closed. Force is generated inside the balloon. This is phase 2. Now mentally, squeeze the balloon with your other hand and let go of the end of the balloon. All the air rushes out rapidly and forcefully. This is phase 3.

Phase 3: Ventricular ejection (points D, E, and F; segment DF). At the exact time that the pressure in the ventricle exceeds the pressure in the recoiling aorta, the **aortic valve opens,** and the **blood rushes out**. The blood is under extreme pressure and so is ejected forcefully through the newly opened valve. Blood is driven forward, down the arterial tree, and into the aorta, distending it to store elastic energy that will be released as the aorta recoils during diastole. The ventricle doesn't suddenly stop generating force when the aorta opens. In fact, the ventricular contraction continues after the aortic valve opens, continuing to eject blood. **Time is not depicted** on a pressure-volume loop. At some point during ventricular ejection, relaxation begins. With the active contraction of sarcomeres ending, the pressure in the ventricle begins to fall. The aorta starts to recoil, pushing blood both down the arterial tree and back into the ventricle. When the pressure in the recoiling aorta exceeds that of the relaxing ventricle, the **aortic valve closes**, **generating S_2.** The aortic valve also closes when no more blood will be expelled, the end-systolic volume. This is the volume where the pressure loop story began—the same as the start of ventricular filling. The difference between EDV and ESV is the stroke volume (SV, how much blood was ejected during ventricular ejection).

Phase 4: Isovolumetric relaxation (points F and A; segment FA). The aortic valve and the mitral valve are both closed, so **no volume change is possible**. The volume in the ventricle is the **end-systolic volume**. The ventricle continues to relax, releasing the actin and myosin cross-bridges, the sarcomeres relaxing. **The pressure continues to fall,** but **no change in volume** happens. As soon as the intraventricular pressure drops below the atrial pressure, the pressure of venous return, the mitral valve opens, and the filling phase begins.

Cardiac Cycle as Seen by the Atrium—and Seen by Venous Pulsation Waves

Whereas the ventricle pressure-volume loop tells the story of the ventricles, the jugular venous pulsation wave tells the story of the atria. We don't actually observe real people's venous pulsation to obtain usable information. Finding a jugular pulsation on an obese patient is difficult enough as is. During the timing of systole (on the order of milliseconds), there are three distinguishable wave patterns and two descents. With point-of-care ultrasound and ease of access to specialists and diagnostics, this isn't something we're preparing you to actually use. We're doing this so that you master systole and diastole.

Because you are supposed to observe the venous pulsation changes in someone's neck, and that vein is connected to the superior vena cava and right atrium, this is the story of the cardiac cycle from the right atrium.

Venous pulsation is a phenomenon that is observable with a bedside technique, and that mirrors the pressure changes in the atrium. As the valves open and close, as the atrium contracts and relaxes, the pressure in the internal jugular vein changes. This is plotted with **time** vs. **pressure**.

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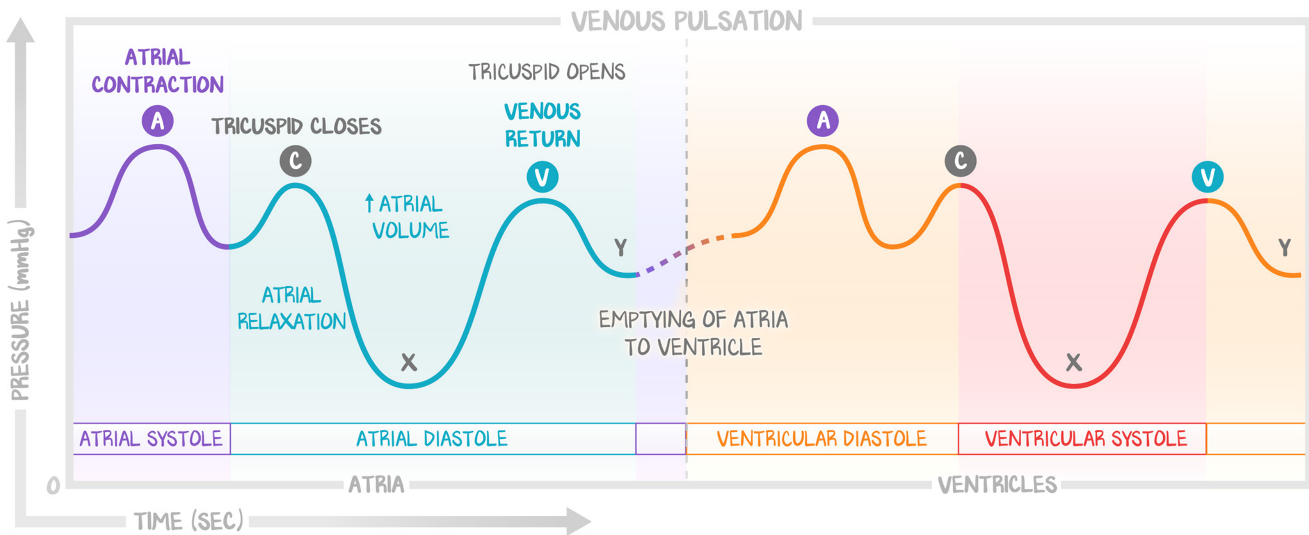

Figure 2.3: Venous Pulsation
Visualization of systole over time using the transient changes in the jugular venous pulsation. The time points are labeled. Atrial systole and diastole are labeled for comparison, on the left, alongside the actions of the tricuspid valve. Ventricular systole and tricuspid actions are layered onto the venous wave on the right. Remember that these are approximations of systole and diastole and that electrical systole and diastole are different from ventricular contraction and relaxation. Use these images to follow along with the text.

Systole begins. The atria depolarize. The atria contract. Being a muscle, the heart's sarcomeres shorten, actin and myosin slide past one another. As the muscle generates tension, the **pressure in the atrium rises**. There are no valves that separate the right atrium from the SVC, so while blood is injected into the ventricle, it is also injected into the SVC and, thus, into the internal jugular vein. The rise in pressure in the internal jugular vein peaks at the ***a* wave** (*a* wave for atrial contraction). The pressure in the walls of the atrium has pushed blood up into the SVC. The pressure in the walls of the atrium has pushed blood into the ventricles, called the **atrial kick**. There is assumed ventricular repolarization and atrial relaxation. The ventricular contraction has not yet been initiated. The tricuspid valve is open.

The atrium begins to relax before the ventricle contracts, a product of the delay through the AV node, delaying the depolarization of the ventricle so that the atrium can deliver its kick to the ventricle. The pressure in the atrium begins to fall. It would fall directly down to the lowest value (**the *x* descent**) because the atrium is simply relaxing, but a major event happens. The ventricle contracts, slamming the tricuspid valve shut.

The tricuspid valve slams shut. If you've ever slammed a cabinet door shut, you may have experienced the cabinet next to it opening. That's because the slamming shut of the cabinet generates force inside the cabinet, and that force can open the door next to it. The slamming of the door shut, the slamming of the valve closed, translates a bit of pressure back into the atrium. So rather than simply falling to the lowest pressure, as would be expected if the atrium relaxed, there is a **sudden uptick in pressure** even though the atrium is no longer contracting. This is the ***c* wave** on the graph (*c* wave for the closure of the tricuspid).

With the tricuspid valve closed and the atrium no longer contracting, the pressure in the atrium drops to its lowest, the ***x* descent** on the graph. It never reaches zero because the venous return is filling the atrium and cannot pass through the tricuspid valve until the right ventricle relaxes. As the volume increases in the right atrium, the walls are passively stretched. That passive stretch creates passive tension, so the pressure increases. The ***v* wave** on the graph (*v* wave for venous return).

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The ventricle relaxes. The pressure in the right atrium exceeds the pressure in the right ventricle. The tricuspid valve opens, and blood flows through. The release of volume from the atrium into the ventricle alleviates that passive tension, and the pressure in the atrium falls. This is the ***y* descent**.

What happens between the *y* descent and the next *a* wave, no one cares about.

Cardiac Cycle as Seen on an ECG

Cardiac conduction and the ECG is discussed in the Electricity island of the Cardiac Module. This isn't meant to be a detailed discussion of the ECG, only to demonstrate electrical systole and diastole as seen from the atria and the ventricles.

The **P wave** is atrial depolarization. The atrial repolarization is lost, buried in the QRS complex. Thus, atrial electrical systole is from the start of the P wave to the start of the QRS complex. Everything else is presumed to be atrial electrical diastole.

The **QRS complex** (in healthy patients, only an RS) marks ventricular **depolarization,** and the **T wave** marks ventricular **repolarization**. From the start of the QRS complex to the end of the T wave is ventricular electrical systole. From the T wave to the next QRS complex is ventricular electrical diastole.

You cannot see the force of contraction, duration of contraction, or opening of the valves; you cannot hear the closure of the valves or visualize a change in volume on an ECG. The amplitude of the waveform is a product of the depolarizing mass of tissue. Is depolarization contraction? Is repolarization relaxation? No. But they are approximated.

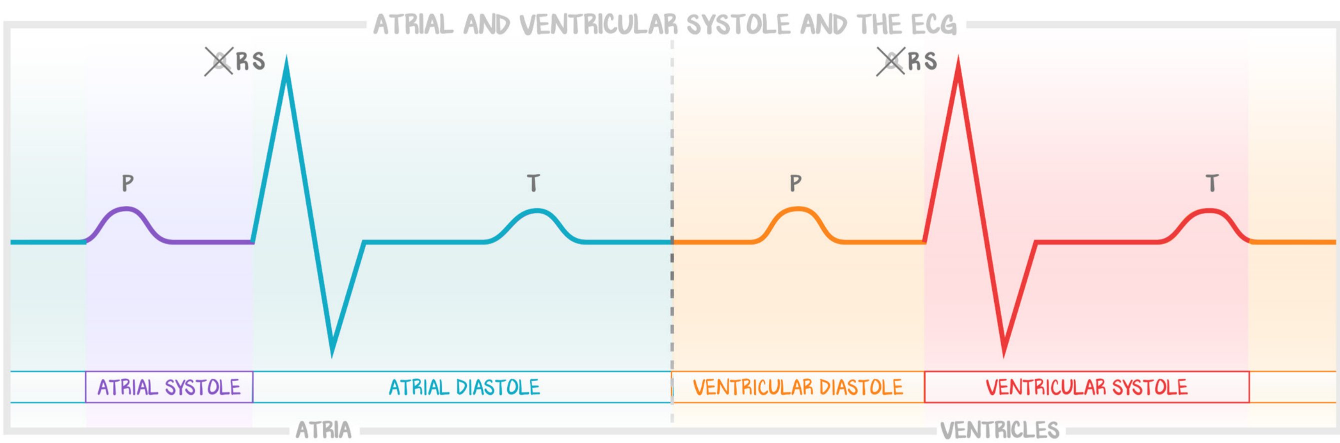

Figure 2.4: Atrial and Ventricular Systole and the ECG
Using the ECG, we illustrate atrial electrical systole and atrial electrical diastole (on the left) and ventricular electrical systole and ventricular electrical diastole (on the right). Q waves are pathologic—a sign of previous myocardial infarction—so are not depicted in our simplified tracings. While the QRS complex is given the name it has, it much more commonly an R (first positive deflection above the line) S (negative deflection that follows the R wave) complex.

The Flow of Blood Through the Heart

If you've been doing the Cardiac module in the recommended order and have been paying attention, then this next section is probably unnecessary. We haven't yet explicitly stated the flow through the circuit, through the two heart model. Because it is a cycle, we can start anywhere.

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The **right heart** receives **deoxygenated blood** from the periphery via three veins: the inferior vena cava, superior vena cava, and cardiac sinus. All of the veins from the inferior part of the body (everything from the extremities and all of the portal system) come together at the **inferior vena cava**. All of the veins from the superior part of the body (arms and head) come together at the **superior vena cava**. All of the cardiac veins come together at the **cardiac sinus**. All three are veins, all three carry **deoxygenated blood**, and all three dump into the **right atrium**. They are separate vessels. They enter the right atrium very close together, but the right atrium is the common drain site.

The right atrium is separated from the right ventricle by the **tricuspid valve** (tricuspid atrioventricular). From the right atrium, blood flows through the tricuspid valve into the **right ventricle**. The right ventricle is not as strong as the left ventricle because it is not as muscular and does not have as thick a myocardium. The pulmonary vascular resistance is significantly less than the systemic vascular resistance—20/10 pulmonary, 120/80 systemic. Deoxygenated blood exits the right ventricle through the **pulmonary valve** (semilunar) into the **pulmonary artery**, which bifurcates, one branch for each lung. The pulmonary arteries carry deoxygenated blood away from the heart towards the lungs, where the blood is then oxygenated.

From the lungs, the **four pulmonary veins** bring **oxygenated blood** back to the **left atrium**. The oxygenated blood passes through the **mitral valve** (bicuspid atrioventricular) into the **left ventricle**. The left ventricle has a thick myocardium. It generates significant force to overcome the systemic vascular resistance. The left ventricular contraction sends oxygenated blood through the **aortic valve** (semilunar) into the **aorta**. The aorta is the main vessel that delivers blood to all its tributaries, distributing oxygenated blood to every organ.

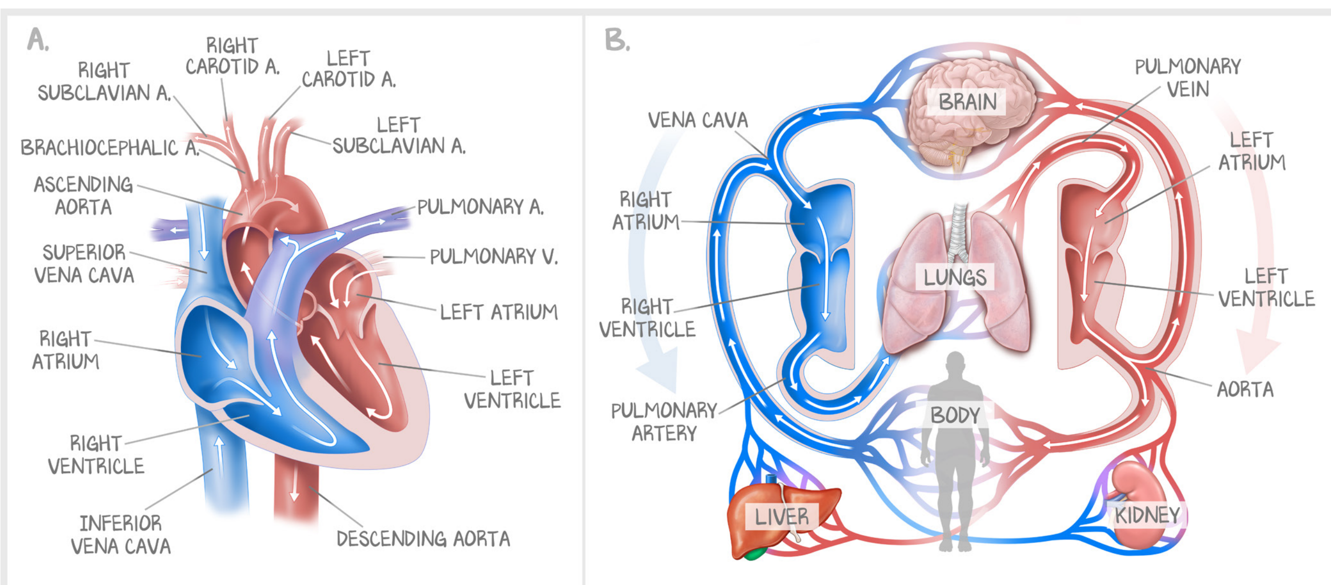

Figure 2.5: Adult Blood Flow
(a) Blood flow as visualized in the real heart. (b) Conceptual flow using the two-heart system. The right heart is deoxygenated and low pressure. Therefore, the right ventricle does not require a thick myocardium. The left heart is oxygenated blood and high pressure. Therefore, the left ventricle requires a thick myocardium.

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Putting It All Together Is a Bad Idea

Depolarization precedes contraction, just like in all muscle. Atrial systole, the initiation of contraction, starts during the P wave. The PR segment allows time to deliver the atrial kick, to increase the end-diastolic volume just a little bit, seen on a venous pulsation curve as the *a* wave. Atrial repolarization is buried in the QRS complex. The atrial pressure heads towards the *x* descent.

Depolarization of the ventricles occurs at the QRS complex. Which ventricles? Both. The contraction will follow close behind. The pressure in the ventricles exceeds that behind the atrioventricular valves, and they close, generating S_1. The *c* wave is visualized on the venous pulsation curve. Isovolumetric contraction begins.

The pressure exceeds that behind the semilunar valves, and blood is ejected. The volume in the ventricles decreases. The opening of the valves makes no noise. There is no electrical change.

Repolarization of the ventricles is the T wave. The ventricles begin to relax until the semilunar valves close, making S_2. The atria now stretch, heading towards their *v* wave. Isovolumetric relaxation begins. The atrioventricular valves open. They make no sound and demonstrate no electrical activity.

If you were confused by the constant changing in modalities, that's okay. If you followed it, then whoa, great for you. It was told as accurately as possible. But there are overlapping events. It isn't one step then the other. It's really close. It's okay to infer. It's okay to put them together. Just recognize that one event in one story very rarely directly corresponds with an event in another.

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